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Modelling of Gas Transport in an Apneic Airway Undergoing Nasal High Flow Therapy

S. Van Hove[1], V. Suresh[1,2] and J. E. Cater[1]

[1]Department of Engineering Science
University of Auckland, Auckland 1010, New Zealand

[2]Auckland Bioengineering Institute
University of Auckland, Auckland 1010, New Zealand

Abstract

Nasal high flow therapy (NHF) has been used during anaesthetic procedures to extend the duration of apnea. It has been observed clinically that the application of the therapy during apnea results in blood O_2 and CO_2 concentrations which remain within a tolerable range for apneic periods lasting up to 14 minutes. However in the absence of ventilation it is unclear how gas transport occurs through the airway during this procedure. A computational fluid dynamics model of the air and CO_2 within the human airway was constructed in order to investigate the transport of gas species during apnea. Flow induced by the beating of the heart was found to be important in the transport and removal of CO_2 from the apneic airway. Application of NHF therapy enhanced the ability of this cardiogenic flow to eliminate CO_2 from the airway. The therapy converts the upper portion of the anatomical dead-space into a supply of fresh gas with low CO_2 concentration and high levels of turbulent kinetic energy. Models which took into account the peripheral airways, using impedance boundary conditions, imposed a lower adverse pressure gradient on the therapy flow. This allows the therapy to washout a greater portion of the anatomical dead-space.

Introduction

Nasal high flow (NHF) therapy delivers heated and humidified gas with a variable oxygen fraction via a nasal cannula to patients with respiratory disorders [4]. In recent years the use of the therapy during anaesthetic apnea has increased in popularity because of its ability to safely extend the time for anaesthetists to intubate patients [6]. Prior to intubation patients are given a neuromuscular blockade thereby inhibiting contraction of the respiratory muscles and preventing spontaneous breathing. Mechanical ventilation can only commence once a successful intubation has been performed. Thus in patients with complicated airway geometries intubation can be a high risk procedure that can result in fatalities.

Patel and Nouraei [6], have clinically demonstrated that application of NHF therapy during anaesthetic apnea allows longer apneic periods while maintaining physiologically acceptable blood gas tensions. In particular the rate of rise of CO_2 in blood was lowered in patients undergoing NHF therapy during apnea compared to rates of rise of CO_2 observed in apneic patients without therapy [6].

The authors attributed the safe extension of apnea to an imbalance in CO_2 and O_2 gas exchange between the airway and blood occurring during apnea [6] that results in a net inflow of roughly $240\,\mathrm{ml\,min^{-1}}$ [1]. While such unidirectional flow is possible, the $4\,\mathrm{ml\,s^{-1}}$ magnitude of this flow is smaller than the oscillatory flow generated by the heart which has been reported to have a root-mean-square tracheal flow of $49 \pm 37\,\mathrm{ml\,s^{-1}}$ [7]. Flow induced by the beating of the heart, or cardiogenic oscillation (COS) as it is referred to, has been suggested to aid gas mixing within the anatomical dead-space [9]. The exact origin of these oscillations is uncertain. *In-vivo* studies have related the pressure and flow waveforms to direct heart-lung contact and/or to the pulsatility of pulmonary blood flow [3, 8].

This work aims to determine the roles cardiogenic oscillations and NHF therapy play in the transport of CO_2 during apnea. As a secondary aim, different methods of modelling the peripheral airway beyond the three-dimensional airway are investigated.

Methodology

An anatomically accurate, patient specific upper airway model was created from a CT scan including the nasal and oral cavities, trachea and the tracheo-bronchiole tree truncated after the seventh branching generation. The small airways beyond the seventh branching generation could not be included in the three-dimensional airway geometry because of the limited resolution of the imaging technique. A tetrahedral mesh was generated along with inflation layers to capture the near-wall flow. The mesh was refined until CO_2 transport was unaffected by further refinement.

The finite volume solver Fluent (ANSYS 18.1) was used to numerically solve the unsteady Reynolds–Averaged Navier–Stokes Equations with a Shear-Stress Transport k-ω turbulence model to close the equations. CO_2 transport was calculated by numerically solving an advection-diffusion equation for the mass fraction of CO_2.

NHF was applied at a flow rate of $70\,\mathrm{l\,min^{-1}}$ resulting in a Reynold number of 10650 at the cannula inlet. In contrast the Reynolds number at the truncated bronchi openings was of the order of 20 due to the small airway diameter and the slow cardiogenic flow passing through these airway vessels. Cardiogenic flow was mimicked by applying a sinusoidal flow waveform to the truncated bronchi outlets of the rigid airway model. The frequency of the oscillations was 60 beats per minute and a peak to peak displaced volume of 20 ml was used. Atmospheric CO_2 concentrations were applied at the cannula inlet boundary and at a zero-gauge pressure region surrounding the exterior of the face. The initial airway CO_2 concentration was equivalent to mean alveolar concentrations (5.3% volume fraction) as was the concentration at the truncated bronchi outlet boundaries.

Two methods were used to account for the absence of peripheral airways. The first was to suspend the airway tree within a constant volume reservoir.

An impedance boundary condition as described by Olufsen and Peskin [5] was applied to the truncated bronchi outlets for the second method. The parameters used to calculate this condition were obtained by fitting the impedance-frequency response to an *in-vivo* response of the entire airway tree published by Chalker, Celli, Habib and Jackson [2]. These parameters were then used to calculate the impedance frequency response of the structured tree extending from each of the truncated bronchi. One-dimensional structured trees were calculated by assum-

ing a power law between parent and daughter branch radii [5]. The structured trees represent the airways beyond the three-dimensional airway geometry. By assuming periodic solutions for pressure and velocity, the linearised one-dimensional Navier-Stokes and continuity equations can be used to calculate the root impedance of a branching vessel network. The impedance frequency response is then used to obtain the time-dependent impedance over a flow oscillation period. The pressure at the truncated bronchi outlets can then be calculated by convolving this impedance with the flow passing through the boundary.

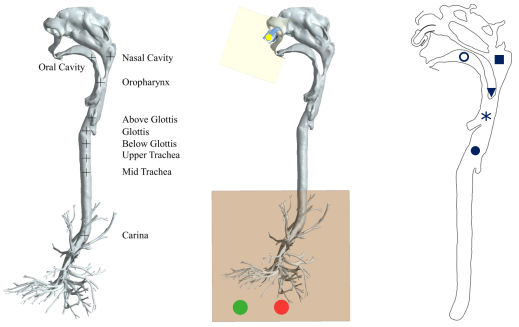


Figure 1: Apneic airway CO_2 sample locations (a), surrounding boundaries (b), sagittal plane with symbols explained in text (c).

The airway geometry is shown in figure 1 with the CO_2 sampling locations (1a), surrounding boundaries including the nasal cannula, atmospheric region around the face and reservoir (1b). A sagittal plane passing through the airway is shown in figure 1c where the symbols indicate the nasal cavity (■), oral cavity (○), uvula (▼), pharynx (∗) and glottis (●).

Results

Four different apneic cases were compared. The first three are from the airway model suspended within the constant volume reservoir and consider the cases where (a) COS is present without NHF, (b) NHF therapy is applied but COS is not and (c) both NHF and COS are present. The last case (d) includes cardiogenic flow and NHF therapy however the impedance boundary condition was applied to the truncated bronchi outlets of this model.

Flow Field

The flow field over a sagittal plane within the airway during peak inspiratory cardiogenic flow is displayed in figure 2. The velocity is plotted with on a logarithmic scale to enhance the visualisation of the differences in flow features within the trachea for each of the airways.

For the case of COS only, figure 2a, the inspiratory flow is laminar throughout the airway and contains no flow recirculations. If cardiogenic flow is absent but NHF therapy is applied, figure 2b, the therapy flow passes through the nasal cavity, down the pharynx and back out the airway via the oral cavity. The majority of the therapy flow attaches to the uvula in response to the expanding nasopharynx. Directly below this some of the therapy flow reaches into the glottis forming two counter rotating recirculation features. However below the glottis region the therapy does not penetrate due to the resistance to flow imposed by the constant volume reservoir.

When NHF is applied and cardiogenic oscillations are present, figure 2c, the corresponding flow field is nearly identical to the linear superposition of the two previous flow fields. This is true for all airway regions except for the glottis region spanning from the uvula to the superior trachea. Within the glottis the recirculating flow induced by the therapy having to rotate 180° is stretched in the inferior direction by the inspiratory cardiogenic flow. This allows the therapy to reach a more inferior location within the airway compared to when cardiogenic oscillations are not present.

With both NHF and cardiogenic oscillations the therapy flow does not attach to the uvula when the peripheral airways beyond the three-dimensional geometry are taken into account with an impedance boundary condition as seen in figure 2d. This differs from the previous case in which the peripheral airways were not considered and the therapy flow attached to the uvula. The impedance boundary condition must impose a smaller adverse pressure gradient on the therapy flow as it passes from the nasopharynx to the oropharynx. This allows the flow to remain attached to the posterior glottis surface until the epiglottis thereby washing out a greater portion of the anatomical dead-space.

Turbulent Kinetic Energy

Figure 3 displays the turbulent kinetic energy within each apneic airway model during peak inspiratory cardiogenic flow. In the absence of NHF therapy, figure 3a the turbulent kinetic energy is low throughout the majority of the airway. Within the oral cavity the turbulent kinetic energy is elevated as the inspired gas from the external atmospheric region enters the airway. However this kinetic energy is quickly damped out by the airway surface resulting in laminar flow within the trachea.

All apneic airway models in which NHF therapy was applied have higher turbulent kinetic energy within the glottis, nasal and oral cavities. This region of high turbulent kinetic energy does not enter the trachea in the absence of cardiogenic flow as seen by the laminar tracheal flow in figure 3b. In figures 3c and 3d the cardiogenic flow permits a higher turbulent kinetic energy within the trachea. Using an impedance boundary condition increased the turbulent kinetic energy all the way to the carina which was not observed in any of the other apneic airway models.

CO_2 Concentration

The normalised CO_2 mass fraction sampled at the six airway locations for each of the four apneic airway models is shown in figure 4.

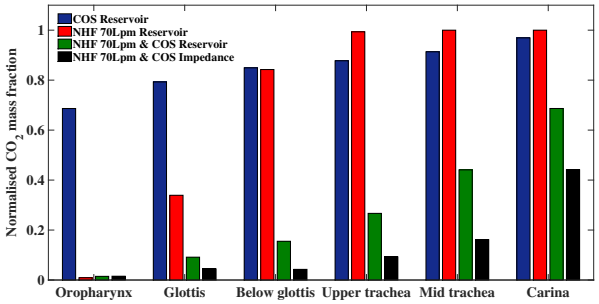
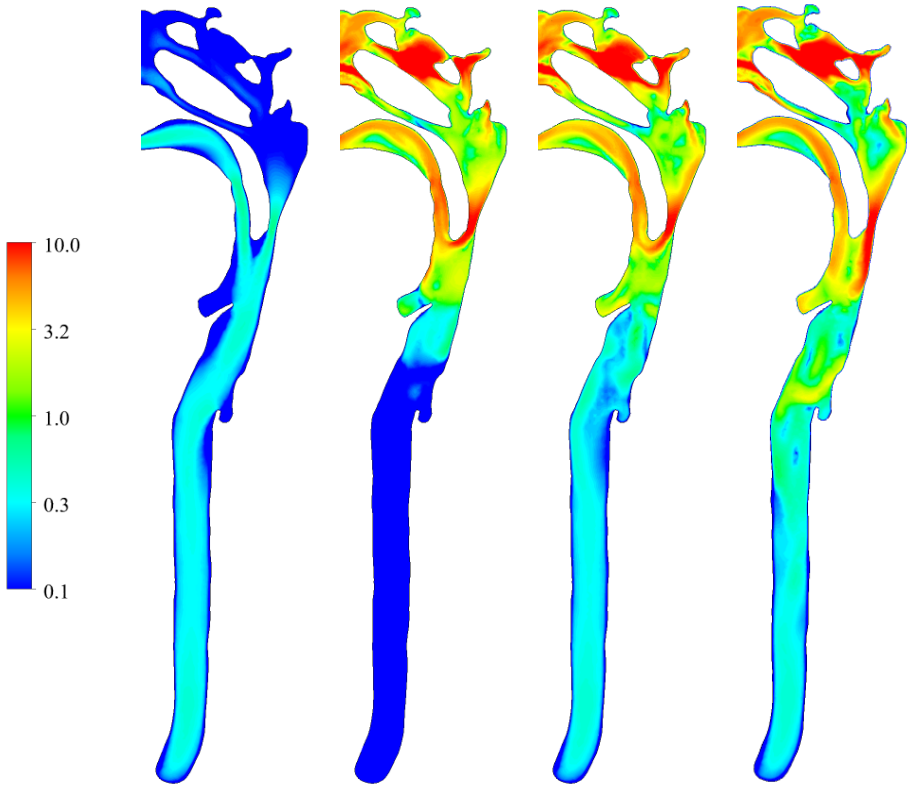


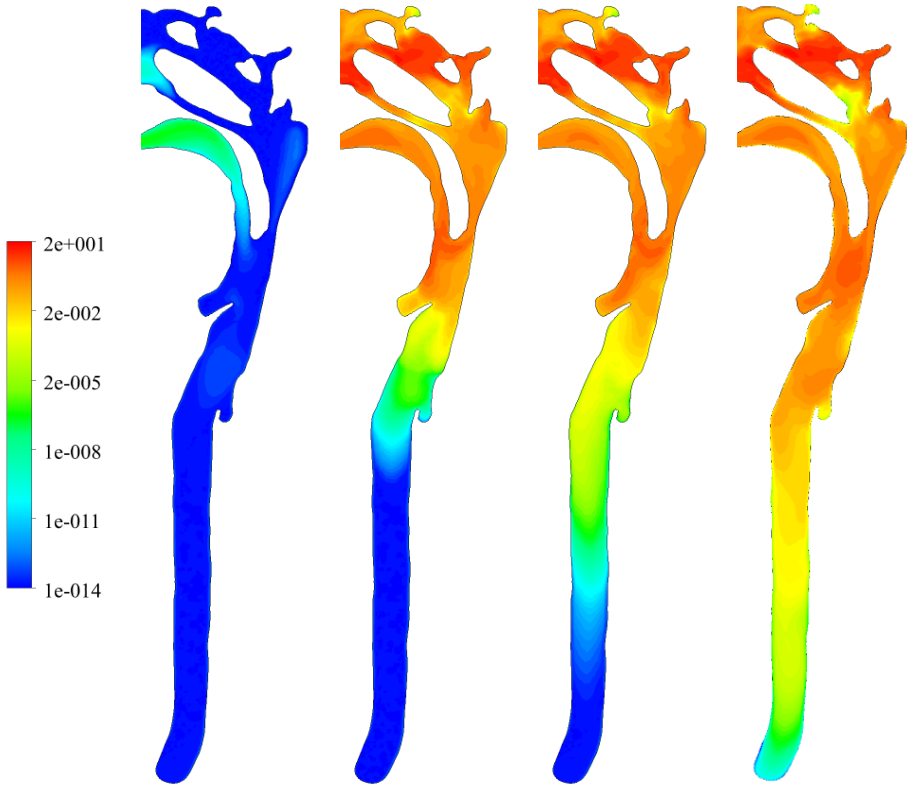
Figure 4: Normalised CO_2 concentration within each of the four apneic airway models after a short apneic period.

CO_2 concentration was normalised by the alveolar concentration and was sampled after ten cardiogenic oscillation. This time-frame was chosen as it is sufficiently long after the initial washout phase, which occurs within the first oscillation period



(a) COS (b) NHF (c) COS & NHF (d) Impedance

Figure 2: Velocity magnitude ($\mathrm{m\,s^{-1}}$) within four apnea airway models during peak inspiratory cardiogenic flow.



(a) COS (b) NHF (c) COS & NHF (d) Impedance

Figure 3: Turbulent kinetic energy ($\mathrm{J\,kg^{-1}}$) within four apnea airway models during peak inspiratory cardiogenic flow.

for the models in which NHF therapy is applied, for a near periodic CO_2 concentration within the airways to be established. The closer the sample location to the oral cavity the lower the CO_2 concentration irrespective of what flow is present within the airway.

For the airway models in which NHF therapy were applied shown by the red, green and black bars, the CO_2 concentration within the oropharynx is equivalent to the atmospheric CO_2 concentration. This is a result of the therapy flow replacing the initial CO_2 rich gas with therapy gas containing a low, or atmospheric, CO_2 concentration. Without cardiogenic flow, red, the CO_2 concentration below the glottis raises to a higher concentration than the concentration in the airway in which cardiogenic flow is present but therapy was not applied blue. This occurs because without cardiogenic flow the therapy flow does not extend past the glottis. Therefore CO_2 transport between the lower airway and the therapy flow in the upper airway is limited by diffusion at the interface between these two regions.

The apneic airway models in which both NHF therapy and cardiogenic flow is present have the lowest CO_2 concentration throughout the airway. When the impedance boundary condition is applied to the truncated bronchi outlets the CO_2 concentration was lower than when the three-dimensional tracheobronchiole tree was suspended in a constant volume reservoir.

Discussion

CO_2 removal from the apneic airways was greatest when both NHF therapy and cardiogenic flow were present. The flow fields and CO_2 concentrations showed that the effect of each of these flows was not a linear combination of the individual effects. This suggests the interaction of these two effects alters the flow field as seen by the unique glottic flow observed when both flows were included in the models.

NHF therapy flushes out upper airway until the glottis. If the therapy flow remains attached to the posterior airway surface as it emerges from the nasopharynx into the oropharynx the extent of flushing by the therapy is increased. Applying impedance based boundary conditions to the truncated bronchi surfaces lowered the adverse pressure gradient within the glottis. This permits the therapy flow to remain attached to the posterior surface instead of attaching to the uvula and therefore washout a greater portion of the airway.

Cardiogenic oscillations provides a conveyor between the lower airway, containing resident or alveolar concentrated gas, and the unidirectional NHF therapy flow passing through the upper airway and out the oral cavity. Without therapy this cardiogenic oscillatory flow is less efficient because the upper airway contains alveolar gas. Replacing this upper airway gas with fresh atmospheric gas will take longer when therapy is absent compared to when therapy is applied. Without cardiogenic flow only the upper airway is flushed by the therapy. Therefore transport of CO_2 between the lower and upper airway is limited by diffusion at the interface between these regions. This interface marks the extent to which the therapy flushes out the airway.

Turbulent kinetic energy is known to enhance heat and mass transfer. Therefore the elevated turbulent kinetic energy, observed when both NHF therapy and cardiogenic flow were included, will have contributed to the increased removal of CO_2 in these apneic airway models compared to those in which only NHF or cardiogenic flows are present. If the impedance boundary conditions are applied to the truncated bronchi outlets the high turbulent kinetic energy from the NHF therapy is able to protrude even more inferiorly in the model than where the airway was suspended within the reservoir.

A lower CO_2 fraction is found within all airway locations in the two models containing both therapy and cardiogenic flow. This is because the interaction between NHF therapy and cardiogenic flow allows a more efficient removal of CO_2 from the airway than with one of these flows alone. The extent to which the therapy reaches inferiorly into the glottis before recirculating determines the amount of the anatomical dead-space flushed out by the therapy. Washout happens nearly instantaneously while a slower removal of CO_2 occurs as the cardiogenic flow conveys gas from the lower airway into the therapy flow stream. This slower removal of CO_2 is faster than diffusion because the cardiogenic flow allows for convective species transport.

Conclusions

Application of NHF therapy enhances the removal of CO_2 concentration from an apneic airway given the presence of cardiogenic flow. Cardiogenic flow is essential for removal of CO_2 in an apneic airway. Using impedance boundary conditions to represent the un-imaged peripheral airways caused the therapy to washout a greater portion of the anatomical dead-space thereby allowing for greater removal of CO_2 from the airway.

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